

MNNR

MORBIDITY AND MORTALITY WEEKLY REPORT

- 741 Outbreaks of Escherichia coli O157:H7 Infection Associated with Eating Alfalfa Sprouts — Michigan and Virginia, June–July 1997
- 744 Silicosis Among Workers Involved in Abrasive Blasting Cleveland, Ohio, 1995
- 748 Nonpolio Enterovirus Surveillance United States, 1993–1996
- 750 Reye Syndrome Ohio, Michigan

Outbreaks of Escherichia coli O157:H7 Infection Associated with Eating Alfalfa Sprouts — Michigan and Virginia, June–July 1997

In June and July 1997, simultaneous outbreaks of *Escherichia coli* O157:H7 infection in Michigan and Virginia were independently associated with eating alfalfa sprouts grown from the same seed lot. The outbreak strains in Michigan and Virginia were indistinguishable by molecular subtyping methods. This report summarizes the preliminary findings of the outbreak investigations.

Michigan

During June 1-July 31, a total of 60 persons with E. coli O157:H7 infection were reported to the Michigan Department of Community Health (MDCH) from 16 counties throughout southern and central Michigan; in comparison, during the same period in 1996, a total of 31 infections were reported throughout the state. Of the 60 persons reported in June and July 1997, isolates from 40 (67%) persons were indistinguishable from each other by pulsed-field gel electrophoresis (PFGE) subtyping performed at the molecular laboratory of MDCH: 14 (23%) had isolates with various other PFGE patterns, and six (10%) had isolates that were unavailable for subtyping. Among the 46 persons whose isolates were indistinguishable by PFGE (40 [87%]) or unavailable for subtyping (six [13%]), the median age was 35 years (range: 2-79 years), and 29 (63%) were female. Bloody diarrhea was reported by 44 (96%) persons, and 25 (54%) were hospitalized. Two persons developed hemolytic uremic syndrome, and one had thrombotic thrombocytopenic purpura; no one died. To assess potential risk factors for infection, a case-control study of 30 case-patients with either the outbreak strain or whose isolates were unavailable was performed, using two age-, sex-, and telephone exchange-matched controls per case; the remaining case-patients could not be contacted to participate, were the second case in a household, or were reported after the study had begun. Of 30 case-patients, 18 (60%) reported eating alfalfa sprouts within 7 days of illness onset, compared with three (5%) of 59 controls during a similar time (matched odds ratio [MOR]=32.1; 95% confidence interval [CI]=5.8-678). No other food items were significantly associated with illness.

All implicated alfalfa sprouts were produced by a single sprouter, who supplied approximately 30%–50% of the alfalfa sprouts marketed in Michigan during the outbreak period. Inspection of the sprouting facility did not identify unsanitary sprout-

E. coli 0157:H7 - Continued

manufacturing practices. Environmental swabs from this grower's facility did not yield *E. coli* O157:H7; microbiologic examination of seed samples is under way.

Virginia

During June 1–July 28, the Virginia Department of Health received reports of 48 cases of *E. coli* O157:H7 infection. In comparison, during the same period in 1996, a total of 20 infections were reported throughout the state. Of the 48 persons reported, 26 (54%) had isolates available. PFGE analysis of these isolates at CDC found that 24 (92%) had indistinguishable PFGE patterns. Among these 24 persons, the median age was 31 years (range: 6–67 years), and 13 (54%) were female. Bloody diarrhea was reported for all persons, and 11 (46%) were hospitalized. To assess potential risk factors for infection, a case-control study of 20 case-patients with the outbreak strain was performed, using one, two, or three age-, sex-, and telephone exchange-matched controls per case. Thirteen (68%) of 19 case-patients with definite responses reported either definitely or probably eating alfalfa sprouts within 7 days of illness onset, compared with six (13%) of 45 controls during a similar time (MOR=24.6; 95% Cl=4.1–537). No other food items were significantly associated with illness.

All implicated alfalfa sprouts were produced by a single sprouter. Inspection of the sprouting facility did not identify obvious unsanitary sprout-manufacturing practices. Environmental swabs from this facility also did not yield *E. coli* O157:H7; microbiologic examination of seed samples is pending.

E. coli O157:H7 isolates from Michigan and Virginia were compared at CDC. Outbreak-associated isolates from both states had indistinguishable PFGE patterns and were phage type 32.

Follow-Up Investigation

In Michigan, implicated sprouts were grown from either of two seed lots that were used interchangeably. In Virginia, implicated sprouts were grown from only one lot, and this lot was the same as one of the lots used in Michigan. The seed lot common to both sprouters was supplied by one seed distributor and was sent to three sprouting facilities; two were the implicated sprouters in Michigan and Virginia, and the third, also in Michigan, reportedly had sprouted only a small amount of the seeds. No tracebacks implicated the third sprouter, none of the sprouts from this sprouter remained on the market, and the remaining seeds from this lot were returned to the distributor. At the Michigan facility implicated by traceback, no sprouts grown from this lot remained on the market, and no seed remained from this lot. In Virginia, the sprouter was still using seeds from the implicated lot; a voluntary recall of sprouts grown from this facility was initiated on August 1, and the remaining seeds were returned to the distributor. No other seed remained unaccounted for from the implicated lot. The mode of contamination of the alfalfa seeds is being investigated.

Reported by: K Como-Sabetti, MPH, S Reagan, Kent County Health Dept, Grand Rapids; S Allaire, Ingham County Health Dept, Lansing; K Parrott, Livingston County Health Dept, Howell; CM Simonds, Washtenaw County Health Dept, Ypsilanti; S Hraboww, MPH, Kalamazoc B Ritter, Midland County Health Dept, Midland; W Hall, MD, J Altamirano, MD, R Martin, PhD, F Downes, PhD, G Jennings, R Barrie, MPH, MF Dorman, N Keon, M Kucab, MPH, A Al Shab, MPH, B Robinson-Dunn, PhD, S Dietrich, L Moshur, L Reese, J Smith, K Wilcox, MD, State Epidemiologist, Michigan Dept of Community Health; J Tilden, DVM, G Wojtala, JD Park, MPH, Michigan Dept of Agriculture. M Winnett, MD, L Petrilack, Medical College of Virginia, Richmond; district health depts; L Vasquez, MSN, S Jenkins, VMD,

E. coli 0157:H7 - Continued

E Barrett, DMD, M Linn, MURP, D Woolard, PhD, R Hackler, H Martin, PhD, D McWilliams, MPH, B Rouse, S Willis, MSW, J Rullan, MD, G Miller, Jr, MD, State Epidemiologist, Virginia Dept of Health; S Henderson, J Pearson, DrPh, Div of Consolidated Laboratory Svcs, Commonwealth of Virginia; J Beers, R Davis, D Saunders, Virginia Dept of Agriculture and Consumer Svcs. Food and Drug Administration. State Br, Div of Applied Public Health Training (proposed), and Div of Prevention Research and Analytic Methods (proposed), Epidemiology Program Office; Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: These are the first reported outbreaks of *E. coli* O157:H7 infection associated with eating alfalfa sprouts. Since 1995, four outbreaks of *Salmonella* infection have occurred in the United States because of consumption of contaminated alfalfa sprouts (1,2; CDC, unpublished data, 1997). In 1996 in Japan, radish sprouts were associated with the largest recorded outbreak of *E. coli* O157:H7 infection, in which approximately 6000 cases occurred (3).

As in previous alfalfa sprout-associated outbreaks of infection with Salmonella serotype Stanley (1) and Salmonella serotype Newport (2), the Michigan and Virginia outbreaks of E. coli O157:H7 infection probably were caused by contaminated alfalfa seeds, rather than contamination during the sprouting process. Because alfalfa seeds are a raw agricultural commodity, they can become contaminated with animal feces that may harbor pathogens such as Salmonella or E. coli O157:H7 during growth, harvest, processing, storage, shipping, or sprouting. The recurrent implication of alfalfa sprouts as a vehicle for foodborne illness highlights the need for strengthened prevention and control measures to ensure the safety of this product. Studies of alfalfa seed inoculated with low numbers of Salmonella suggest that the number of organisms present on seeds may increase up to 10,000-fold during the sprouting process (4). The effect of the sprouting process on the growth of E. coli O157:H7 is unknown. In April 1996, representatives of the sprout industry met with the Food and Drug Administration (FDA) and CDC to discuss research needs to identify effective methods of alfalfa seed decontamination. However, research has not identified such a method; treatments, such as soaking seeds in water with chlorine concentrations of 2000 ppm (the highest allowable concentration), reduce the level of contamination but can leave viable microorganisms that may then be amplified during the sprouting process (4). Further efforts are needed to evaluate seed and sprout disinfection strategies.

In addition to seed decontamination, prevention of future alfalfa sprout-related outbreaks will depend on identification of critical control points to reduce the likelihood of contamination during seed production and distribution. Additional prevention approaches could include categorizing sprout growers as food service workers rather than agricultural harvesters, along with systematic inspection and certification of sprouting facilities. As with all fresh produce, consumers should thoroughly rinse alfalfa sprouts before eating; however, the effectiveness of rinsing to reduce contamination is unknown. Persons at higher risk for severe complications of *E. coli* O157:H7 or *Salmonella* infection, such as infants and young children, the elderly, pregnant women, or immunocompromised persons, can reduce their risk by not eating raw alfalfa sprouts.

The Michigan and Virginia E. coli O157:H7 outbreaks demonstrate the value of molecular subtyping in the investigation of foodborne outbreaks. In both states, an increase in the number of reported cases of E. coli O157:H7 infection was suggested by

E. coli O157:H7 - Continued

PFGE analysis to be a common-source outbreak rather than an increase in sporadic cases. In addition, molecular subtyping of isolates from both states by PFGE and phage typing at CDC demonstrated that these outbreaks were linked by a common strain, corroborating the epidemiologic and traceback findings. CDC has established a National Network for Molecular Subtyping (5), with four area laboratories in Massachusetts, Minnesota, Texas, and Washington serving as reference PFGE laboratories; other state laboratories also have begun using the same method. Standardized laboratory procedures and electronic links to share data among laboratories and CDC make this network a key element of the recently announced President's Food Safety Initiative (6) and an important aspect of outbreak detection and coordination.

References

- Mahon BE, Ponka A, Hall WN, et al. An outbreak of Salmonella infections caused by alfalfa sprouts grown from contaminated seeds. J Infect Dis 1997;175:876–2.
- Van Beneden CA, Keene WE, Werker DH, et al. A health food fights back: an international outbreak of Salmonella Newport infections due to alfalfa sprouts. In: Program and abstracts of the 36th Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1996.
- Infectious Disease Surveillance Center. Verotoxin-producing Escherichia coli (enterohemorrhagic E. coli) infections, Japan, 1996–June 1997. IASR (Tokyo, Japan) 1997;18:153–4.
- Jaquette CB, Beuchat LR, Mahon BE. Efficacy of chlorine and heat treatment in killing Salmonella Stanley inoculated onto alfalfa seeds and growth and survival of the pathogen during sprouting and storage. Applied and Environmental Microbiology 1996;62:2212–5.
- Stephenson J. New approaches for detecting and curtailing foodborne microbial infections. JAMA 1997:277:1337–40.
- 6. US Environmental Protection Agency/US Department of Health and Human Services/US Department of Agriculture. Food safety from farm to table: a national food-safety initiative—a report to the President, May 1997. Washington, DC: US Environmental Protection Agency/US Department of Health and Human Services/US Department of Agriculture, 1997.

Silicosis Among Workers Involved in Abrasive Blasting — Cleveland, Ohio, 1995

Silicosis is a debilitating lung disease caused by inhalation of crystalline silica. An estimated 2 million U.S. workers are at risk for silicosis (1); approximately 100,000 of these work as sandblasters. On April 21, 1995, CDC's National Institute for Occupational Safety and Health (NIOSH) received a technical assistance request from the Ohio Department of Health (ODH) to conduct medical screening of Cleveland area workers involved in or around abrasive blasting activities. The request was based on the identification of high exposures to crystalline silica and deficiencies in the respiratory protection program at a worksite where an employee who worked as an abrasive blaster had died with accelerated silicosis in 1992 (2). This report summarizes the results of the survey conducted in response to this request, which identified eight workers with radiographic evidence of pneumoconiosis and indicated that inappropriate selection and use of respirators during abrasive blasting operations were common.

The objectives of the survey were to identify workers with silicosis and to assess the workers' knowledge about proper use of respiratory protection and appropriate industrial hygiene practices for the prevention of exposures to silica. Participants were Silicosis -- Continued

recruited from the membership rosters of a trade union, through the Cleveland office of the ODH, and through media advertisements. Screening was performed by NIOSH investigators during August 7–11 and August 21–25, 1995, in the metropolitan Cleveland area.

Screening consisted of a work history, a medical questionnaire, and a chest radiograph. Occupational questions focused on the intensity and characteristics of exposures to silica. Evaluation of workers' knowledge and the adequacy of respiratory protection programs was based on the NIOSH Respirator Decision Logic (3) and the Occupational Safety and Health Administration (OSHA) regulations*. Radiographs were sent for independent readings by two NIOSH-certified B readers (physicians trained and certified in the classification of chest radiographs for pneumoconioses) who, without knowledge of the participant's age, occupation, or smoking history, classified the films according to the international classification system for pneumoconiosis (4). A case of silicosis was defined as a chest radiograph with an International Labor Organization (ILO) classification of ≥1/0 (4) in a worker with a history of exposure to silica dust. No exposure assessments or worksite visits were performed.

Of 170 participants, 122 (72%) were employed, and 48 (28%) were unemployed (including retired or disabled persons). Most were male (166 [98%]), white (160 [94%]), and non-Hispanic (166 [98%]). The median age was 48 years (range: 24–78 years). The prevalence of current smoking was 37%; 42% were former smokers.

Three fourths of participants described their usual job title as painter (62%) or dry-wall finisher (13%). Eighty (47%) reported ever having performed blasting; the median number of years these workers had performed blasting duties was 11 (range: 1–45 years). Ninety-six (56%) participants had ever worked as a blaster's helper (median tenure: 8 years), and 63 (37%) reported performing both blasting and blaster's helper duties.

Among the 122 employed workers, 47 (39%) had duties directly related to blasting—11 (9%) were exclusively blasters, 13 (11%) were helpers, and 23 (19%) performed both activities. Of these 47, a total of 43 (92%) reported that their employers did not require them to use respirators at their current jobs. However, only one employee reported that his employer did not provide respirators, and 43 (92%) workers reported using a respirator while performing their duties. Thirty-one (66%) reported that their current employer had informed them about the health hazards of sand or silica dust, and the same number indicated that their current employer had given them training in the use of a respirator.

Among the 34 workers directly performing blasting (i.e., blasters and those who worked both as blasters and as helpers), 11 (32%) reported using an air-supplied hood with tight-fitting face piece, but only two had been fit-tested for this respirator. A total of 27 (79%) reported using a replaceable-cartridge air-purifying respirator (10 [37%] of these had been fit-tested), and six (18%) reported using a dust mask while operating

^{*29} CFR 1926.103.

^{*}Blaster's helpers are apprentices to workers involved in abrasive blasting and are required to keep the blasting pot filled with abrasive and to oversee other activities directly related to the abrasive blasting process (e.g., the functioning of the air compressor). Because of their proximity to blasting operations, they also are at risk for high exposure to silica dust.

Silicosis — Continued

blasting equipment. In addition, 12 used a dust mask when working around blasters and 17 when cleaning up.§ Eight (24%) of the 34 had interfering facial hair.§

Silica sand was the most commonly used abrasive (21 [62%] of the 34 workers used this abrasive most often). Other frequently used abrasive materials included steel shot and coal slag.

Of the 113 survey participants who had ever blasted or worked as helpers, eight (7%) had chest radiographs that were classified as being consistent with pneumoconiosis; all eight were classified as major profusion category one or greater (ILO classification ≥1/0). All were men; their median age was 63 years (range: 41–71 years). Two (25%) were current smokers, and three (38%) were former smokers. Only one of these eight had known that his chest radiograph showed evidence of pneumoconiosis. All eight had performed blasting**; half were employed, but only one was currently blasting.

Reported by: A Migliozzi, MSN, K Gromen, Ohio Dept of Health. Clinical Investigations Br, Div of Respiratory Disease Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Silicosis is a disabling and potentially fatal lung disease that has no cure but is completely preventable by controlling exposures to respirable crystalline silica. Identification of silicosis in a worker is considered a sentinel event and should prompt an investigation of workplace exposures and work practices. NIOSH has published reporting guidelines and a surveillance case definition for silicosis (5), and seven states conduct surveillance for silicosis (6).

The findings in this report highlight continued lack of knowledge about proper selection and use of respirators and other silicosis-prevention practices among workers in occupations with exposures to crystalline silica. The only respirators suitable for use in abrasive blasting operations are type CE abrasive-blast supplied-air respirators; air-purifying and powered-air purifying respirators are not recommended for abrasive blasting operations but may be suitable for auxiliary work (e.g., outside clean-up operations). In this report, only 11 (32%) of the 34 workers directly involved in blasting activities reported using an air-supplied hood with a tight-fitting face-piece respirator, an appropriate respirator for these workplace circumstances, and most of these 11 had not been fit-tested for this respirator. As a result, even workers who had selected the type of respirator recommended for their duties may not have been using their respirators effectively (all respirators must be properly fit-tested when initially assigned to a user). Of particular concern is the reported use of simple dust masks in conjunction with blasting. Finally, approximately one fourth of survey participants who were directly involved in blasting activities had interfering facial hair and would not have had the proper face-to-respirator seal necessary for adequate protection. NIOSH recently updated information about recommendations for selection and use of respirators by workers involved in abrasive blasting (7).

Respiratory protection is considered supplementary to dust source controls at the workplace; NIOSH has recommended that silica sand or other substances containing

Some workers reported using more than one type of respirator or using different types of respirators for different activities.

Facial hair that lies along the sealing area of a respirator and may interfere with the face-torespirator seal.

e*Only one of the eight had a history of other occupational exposures in which high exposures to silica could have led to silicosis; this worker had worked in a coal mine for 3 years, in a foundry for 2 years, and in a glass factory for 3 years.

Silicosis — Continued

more than 1% crystalline silica should not be used as blasting abrasives and that less hazardous materials should be substituted. In blasting activities using silica sand as the abrasive material, the concentrations of airborne dust containing respirable silica can vary considerably but are generally well above recommended safe levels. The results of chest radiograph screening and work practice questionnaires among survey participants indicate that a potential health hazard exists in these workers and that they may be at risk for developing silicosis.

The findings in this report are subject to at least three limitations. First, a large portion of the high-risk group was not evaluated (the union surveyed for this report has approximately 1500 members, and an additional 500 to 1000 nonunionized area workers are engaged in abrasive blasting). Second, survey participants may not have been representative of all workers engaged in abrasive blasting in the Cleveland area. Therefore, the observed results may underestimate the extent of the problem in the Cleveland area, and the degree to which these results may reflect conditions in other regions is uncertain. Finally, no worksite assessments or environmental samplings were performed as part of this study. Nevertheless, the survey identified unrecognized cases of pneumoconiosis, and the findings underscore a lack of knowledge about potential exposures to silica dust and good workplace practices among workers engaged in high-risk occupations such as abrasive blasting.

To better inform both employers and workers about the hazards of silica dust exposure and about good work practices for silicosis prevention, OSHA implemented the Special Emphasis Program for Silicosis Prevention within the state of Ohio. Outreach activities began in June 1996, followed by enforcement activities in July. Activities to educate workers exposed to airborne crystalline silica are continuing through the efforts of the OSHA regional office in Cleveland. NIOSH has published an alert requesting assistance in preventing deaths among sandblasters (8).

References

- National Institute for Occupational Safety and Health. Work-related lung disease surveillance report, 1994. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, CDC, 1994; DHHS publication no. (NIOSH)94-120.
- National Institute for Occupational Safety and Health. Hazard evaluation and technical assistance report: commercial steel treating company, Cleveland, OH. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, CDC, 1992; NIOSH report HETA 92-0174.
- National Institute for Occupational Safety and Health. NIOSH respirator decision logic. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, CDC, 1992; DHHS publication no. (NIOSH)87-108.
- International Labor Organization. Guidelines for the use of ILO international classification of radiographs of pneumoconioses. Geneva, Switzerland: International Labor Organization, 1980.
- CDC. Silicosis: cluster in sandblasters—Texas, and occupational surveillance for silicosis. MMWR 1990;39:433–7.
- Maxfield R, Alo C, Reilly MJ, et al. Surveillance for silicosis, 1993—Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin. In: CDC surveillance summaries (January) MMWR 1997;46(no. SS-1):13–28.
- National Institute for Occupational Safety and Health. NIOSH respirator users notice. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, CDC, May 23, 1996.
- National Institute for Occupational Safety and Health. NIOSH alert: preventing silicosis and deaths from sandblasting. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, CDC, 1992; DHHS publication no. (NIOSH)92-102.

Nonpolio Enterovirus Surveillance — United States, 1993-1996

Each year in the United States, an estimated 30 million nonpolio enterovirus infections cause aseptic meningitis; hand-foot-and-mouth disease; and nonspecific upperrespiratory disease (1). From January 1993 through December 1996, state public health laboratories reported to CDC virus isolation results for 3209 specimens tested for nonpolio enteroviruses (Table 1). The number of states reporting enterovirus isolations decreased from 25 in 1993 to 14 in 1996. This report summarizes surveillance data for nonpolio enteroviruses and describes temporal trends in nonpolio enterovirus infections during 1993–1996.

During 1993–1996, of the 3209 nonpolio enterovirus isolations reported, echovirus 9 was the predominant serotype reported (12.7%), followed by coxsackievirus B5 (11.5%), echovirus 30 (9.5%), coxsackievirus A9 (6.6%), coxsackievirus B2 (6.2%), echovirus 6 (5.1%), and echovirus 11 (4.5%). None of the 67 known enterovirus serotypes was listed in 3.8% of the reports. Isolates were most frequently obtained from cerebrospinal fluid (25.3%), nasopharyngeal swab (17.6%), and stool (14.1%) specimens.

For the patients from whom enteroviruses were isolated, provisional clinical diagnoses included aseptic meningitis (12.9% of patients), encephalitis (3.7%), pneumonia or respiratory symptoms (3.1%), paralysis (0.03%), and carditis (0.03%). No clinical diagnosis was noted for 72.9% of patients for whom specimens were submitted. Reported by: State virology laboratory directors. Respiratory and Enterovirus Br, Div of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: To assess temporal trends in enterovirus infections, public health laboratories have voluntarily reported enterovirus isolations by serotype to CDC since 1951. Surveillance data have consistently demonstrated seasonal peaks in enterovirus isolations during summer and early fall (2). Although the predominant enterovirus serotype reported varies each year, certain serotypes are among the most commonly detected each year (1-3). Surveillance data for 1993-1996 are consistent with these previous observations. Excluding the reports with "unknown" serotypes, six serotypes (echovirus 6, 7, 11, and 30, and coxsackievirus A9 and B4) were among the 10 most frequently detected serotypes each year, and echovirus 9 and coxsackievirus B5 were among the 10 most frequently detected serotypes for 3 of the 4 years. During the 4-year period, the 10 most frequently reported serotypes (the above eight serotypes plus coxsackievirus B2 and coxsackievirus B3) accounted for 58.3%-80.8% of isolates each year. Nine of these 10 serotypes also were among the 10 most common serotypes isolated during 1970-1983-the period on which the last enterovirus surveillance report was based (2,3). Echovirus 4 was not among the 15 most frequently isolated serotypes reported during 1993-1996; however, it was the fifth most common serotype reported during 1970-1983. Echovirus 7 was the eighth most common serotype isolated during 1993-1996 and the 12th most common serotype isolated during 1970-1983.

The findings in this report are subject to at least two limitations. First, these nationwide aggregated surveillance data are the only information available to describe the major temporal trends in enterovirus infections in the United States; however, these data may not be representative of the general U.S. population. Second, the impact of

Nonpolio Enterovirus Surveillance — Continued

	1993		1994		1995		1996		1993-1996	96
Rank	Virus type	%								
-	ECHO 30	25.9%	Coxsackie B2	16.5%	ECHO 9	45.1%	Coxsackie B5	31.6%	ECHO 9	12.7%
2	Coxsackie B5	8.5%	Coxsackie B3	10.5%	ECHO 11	6.8%	ECHO 17	10.6%	Coxsackie B5	11.5%
3	Coxsackie A9	7.6%	Unknown	10.1%	Coxsackie A9	5.7%	ECHO 6	9.4%	ECHO 30	9.5%
4	ECHO 7	5.8%	ECHO 6	7.0%	Coxsackie B2	5.3%	Coxsackie A9	8.6%	Coxsackie A9	6.6%
2	Coxsackie B3	4.6%	ECHO 30	2.9%	Coxsackie B5	4.4%	Coxsackie B4	7.7%	Coxsackie B2	6.2%
9	Coxsackie B4	3.8%	Entero 71	2.6%	ECHO 30	4.4%	ECHO 11	4.4%	ECHO 6	5.1%
7	ECHO 25	3.8%	Coxsackie A9	4.4%	ECHO 7	4.1%	ECHO 7	4.4%	ECHO 11	4.5%
00	ECHO 11	3.1%	ECHO 11	3.8%	Coxsackie B4	3.3%	Unknown	3.2%	ECHO 7	4.4%
6	ECHO 6	2.7%	ECHO 7	3.3%	ECHO 25	1.9%	ECHO 22	3.2%	Coxsackie B4	4.4%
10	ECHO 22	1.8%	ECHO 9	2.8%	Unknown	1.6%	ECHO 9	2.4%	Coxsackie B3	4.0%
11	Coxsackie B2	1.8%	Coxsackie B4	2.8%	ECHO 6	1.2%	ECHO 30	1.8%	Unknown	3.8%
12	Entero 71	0.7%	ECHO 25	2.8%	ECHO 22	1.1%	ECHO 25	1.5%	ECHO 17	2.7%
3	ECHO 9	0.7%	ECHO 22	2.8%	Entero 71	%9.0	Entero 71	1.5%	ECHO 25	2.5%
14	Unknown	0.1%	Coxsackie B5	1.5%	Coxsackie B3	0.4%	Coxsackie B2	1.2%	ECHO 22	2.2%
15	ECHO 17	0.1%	ECHO 17	0	ECHO 17	0	Coxsackie B3	0.3%	Entero 71	2.1%
Total	Total number									
of is	of isolates	1357		612		901		339		3209

Nonpolio Enterovirus Surveillance - Continued

the decreasing number of laboratories reporting enterovirus isolations to CDC on the capacity to describe temporal trends cannot be determined.

Despite these limitations, these data reemphasize two points about enterovirus infections. First, annual variability in the predominant enterovirus serotypes must be considered in studies of possible links between enterovirus infections and disease. Second, the consistent presence of certain serotypes among the 10 most frequently detected enterovirus serotypes can be used to focus diagnostic activities in virology laboratories.

References

- Morens DM, Pallansch MA, Moore M. Polioviruses and other enteroviruses. In: Belshe RB, ed. Textbook of human virology. 2nd ed. St. Louis, Missouri: Mosby, 1991:427–97.
- Strikas RA, Anderson L, Parker RA. Temporal and geographic patterns of isolates of nonpolio enterovirus in the United States. 1970–1983. J Infect Dis 1986:153:346–51.
- 3. Moore M. Enteroviral disease in the United States, 1970-1979. J Infect Dis 1982;146:103-8.

As part of its commemoration of CDC's 50th anniversary, MMWR is reprinting selected MMWR articles of historical importance to public health, accompanied by current editorial notes. Reprinted below is the report published November 7, 1980, that presents findings of two studies describing the association between Reye syndrome and aspirin.

Reye Syndrome — Ohio, Michigan

In addition to a previously reported study from Arizona (1), CDC has received reports of studies conducted in Ohio and Michigan which suggest a relationship between Reye syndrome and salicylates (i.e., aspirin) taken during an associated antecedent illness.

Between December 1978 and March 1980, a prospective case-control study of Reye syndrome was conducted by the Ohio State Department of Health. This study involved 6 pediatric centers in the state and examined the possible relationship between Reye syndrome and medications taken during the antecedent illness. One hundred fifty-nine cases were identified in this study; slightly more than half were relatively mild, developing only stage I encephalopathy (difficult to arouse, lethargic, sleepy). A large percentage of these patients were identified during an outbreak of influenza A (H1N1) that occurred in December 1978-March 1979 and an outbreak of influenza B that occurred in December 1979-March 1980, or had varicella as an antecedent illness.

Reye syndrome patients and controls, selected from the same school classroom or neighborhood and matched for age, sex, race, and the occurrence of a similar antecedent illness (respiratory, varicella, or gastrointestinal) within 1 week of that which occurred in the case, were interviewed concerning medications taken between the time of onset of the antecedent illness and either admission to the hospital for Reye syndrome (for cases) or recovery from the illness (for controls). For each Reye syndrome case, the date of onset of vomiting, which is usually associated with the onset of Reye syndrome, was recorded. The frequency of usage of only 2 medications was found to be significantly different statistically in cases and controls. Salicylates, including those contained in various compounds, were the only medications which

Reye Syndrome — Continued

were taken significantly more frequently in cases (95/98, 97%) than controls (114/160, 71%) (p<.001). All of the Reve syndrome cases with a history of salicylate ingestion took salicylates during their antecedent illness and prior to the onset of the preencephalopathic vomiting associated with this syndrome. Multiple logistic analysis using a model that included histories of salicylate ingestion, fever, headache, and sore throat has demonstrated that although a history of fever was significantly greater in cases than controls, this difference did not account for the even stronger association of cases with a history of salicylate ingestion. Using this model, the estimated relative risk of Reye syndrome for patients taking salicylates was 11.3 (95% confidence limits 2.7-47.5). Histories of headache and sore throats were not significantly different in cases and controls. Medications containing acetaminophen were taken by only 16% (16/98) of cases compared to 32% (51/160) of controls (p<0.01). Although analysis has not yet been completed concerning the dose of salicylates ingested by the patients with Reye syndrome, the majority had a history of taking no more than normally recommended. The medication history was usually obtained from parents within 7-10 days (for cases) and 10-20 days (for controls) after the onset of antecedent illness.

The recently reported study from Michigan involved 25 patients with Reye syndrome and 44 controls selected in a manner similar to that of the Ohio study, matched for the same criteria, and interviewed 4 to 83 days (mean 6.5 weeks) after their acute illness. When cases and controls were retrospectively matched for fever (±1° F), aspirin was taken significantly more often in cases (14/14, 100%) than controls (14/21, 67%, p<0.02), and acetaminophen-containing compounds were taken significantly less often in cases (0/14), than in controls (6/21, 29%, p<.05).

Reported by TJ Halpin, MD, State Epidemiologist, F Holtzhauer, Ohio State Dept of Health; Dept of Epidemiology, University of Michigan School of Public Health; N Hayner, MD, State Epidemiologist, Michigan Dept of Public Health; Field Services Div, Viral Diseases Div, Bur of Epidemiology, CDC.

Editorial Note: Although the epidemiologic association between Reye syndrome and antecedent viral illnesses is well established, the etiology of this rare disease remains unclear. Several previous reports have suggested the possibility that medications taken during the antecedent illness of patients with Reye syndrome may play a role in the development of this disease, and aspirin is 1 medication which has been mentioned frequently (2-4).

The Ohio and Michigan studies reported here and the previously reported smaller study from Arizona (involving 7 cases and 16 controls) are the only controlled studies of the relationship between Reye syndrome and medications taken during the antecedent illness reported since this disease was first described. All 3 of these studies involved in-home interviews focusing specifically on medication histories of Reye syndrome patients and controls.

A number of potential problems are encountered when conducting and analyzing such studies. These include 1) difficulties in obtaining comparable and accurate medication histories in patients following a significant event (Reye syndrome) when compared to controls who have had a relatively minor illness, and the difficulty of accurate recall of events several weeks later, 2) the possibility that cases had a more severe antecedent illness and/or a pre-encephalopathic illness that included severe vomiting and headaches—both of which may have predisposed them to take more medications than controls—and 3) the presumed need to select cases and controls with the same

Reve Syndrome — Continued

viral infections, including influenza B, influenza A (H1N1), and varicella, since Reye syndrome is thought to be more strongly associated with these infections.

It is possible that parents of patients with Reye syndrome were more likely than parents of controls to recall events immediately preceding their child's major illness and hospitalization, including medications taken by their child during this period. Recall of medication histories for Reye syndrome patients may also have been more accurate and complete than the recall for controls because parents of cases were frequently interviewed earlier after their child's acute illness than were parents of controls. However, the fact that only aspirin or salicylate-containing compounds were found to have been taken significantly more frequently during the antecedent illness in cases than controls in these studies suggests that the association between Reye syndrome and salicylates may indeed be real. Furthermore, the fact that acetaminophen-containing compounds were taken by significantly fewer cases than controls in both studies, which might be expected if Reye syndrome patients were more likely to use salicylates than acetaminophen for fever or other symptoms, suggests that the recall of parents of cases was not greater than the recall of parents of controls for these medications.

Another possible reason for differences in medication histories in cases and controls is that Reye syndrome patients may have a more severe or prolonged antecedent illness and/or may subsequently develop a pre-encephalopathic illness, associated with severe vomiting, for which they might receive additional medications. Because elevated temperatures are 1 major reason for taking salicylates, both of these studies have attempted to compare the effects of differing histories of fever among cases and controls. In the Michigan study, even when cases and controls were matched for degree of fever, the difference in salicylate usage remained significant. Analyses completed in the Ohio study have demonstrated that a history of fever, as well as headaches and sore throats-symptoms which might also cause cases to take more salicylates than controls—did not account for the observed differences in salicylate ingestion. Additional analyses in Ohio of aspirin ingestion histories of Reye syndrome patients for the specific period between onset of prodromal illness and onset of vomiting demonstrated that all of 95 patients who received salicylates received some during their antecedent illness-before the onset of pre-encephalopathic vomiting. The possible confounding effects of other symptoms and combinations of symptoms are being further examined in the Ohio study.

Reye syndrome is rare and associated frequently with certain viruses. Thus, comparison of medication histories in cases and controls who had the same viral infection may be important. In both of these studies, controls were selected from the same school and had a prodromal illness within 1 week of that of the cases. It is probable that many cases and controls were matched for infection because a large percentage of the cases occurred during outbreaks of influenza, and varicella patients were matched with other children who had varicella. Further analysis of the salicylate association by specific type of infection should be possible in the Ohio study.

In 1976 the Food and Drug Administration advised that, when treating children who develop vomiting associated with a viral illness, caution should be exercised in using acetaminophen, salicylates, and antiemetics because of the suspicion that these drugs, in combination with a viral illness (a possible cause of vomiting in children) might contribute to the development of Reye syndrome (5). The results of these stud-

Reve Syndrome - Continued

ies suggest that during certain viral illnesses the use of salicylates—even before the onset of vomiting—may be a factor in the pathogenesis of Reye syndrome. In view of these data, parents should be advised to use caution when administering salicylates to treat children with viral illnesses, particularly chickenpox and influenza-like illnesses.

References

- 1. MMWR 1980;29:321-2.
- 2. Giles HM. Encephalopathy and fatty degeneration of the viscera. Lancet 1965;1:1075.
- Linnemann CC, Shea L, Partin JC, Schubert WK, Schiff GM. Reye's syndrome: epidemiologic and viral studies, 1963-1974. Am J Epidemiol 1975;101:517-26.
- DeVivo DC. Reye syndrome: a metabolic response to an acute mitochondrial insult? Neurology 1978;28:105-8.
- 5. FDA Drug Bulletin, Vol. 6, No. 5. Nov-Dec 1976.

Editorial Note—1997: Reye syndrome (RS) was first described in Australia (1) and in the United States in 1963 (2). During the 1960s and 1970s, RS outbreaks in the United States became increasingly recognized in association with outbreaks of influenza and following chickenpox. National surveillance for RS, first conducted during the 1973–74 nationwide epidemic of influenza B, resulted in the recognition of regional as well as nationwide outbreaks of RS. Although children of all ages were affected, incidence peaked among children aged 5–15 years. During the initial years of national surveillance, 236–555 cases were reported each year; the largest number occurred in association with outbreaks of influenza B and influenza A(H1N1). Population-based studies suggested that the average annual incidence among children aged <18 years was approximately one case per 100,000 persons. Case-fatality rates reported through national surveillance were initially as high as 40% and between 20% and 35% during the late 1970s to mid-1980s.

Although anecdotal reports during the 1970s had suggested the possibility of an association between RS and aspirin, the series of studies reported in 1980—the first from Arizona (involving seven cases and 16 controls) followed by larger studies conducted in Michigan and Ohio—were the first case-control studies to examine this issue. However, the possibility that a commonly used medication such as aspirin, which had been prescribed for several decades for febrile illnesses by those taking care of pediatric patients, might be associated with a severe and frequently fatal illness was not readily accepted by many in the medical community. As a reflection of the controversial nature of this matter, the initial Editorial Note published in the November 7, 1980, issue of MMWR outlined several of the most important potential limitations of these studies and the considerations that had led CDC to conclude that the studies were strongly suggestive of an association between aspirin use and increased risk for RS. In an effort to fulfill CDC's public health responsibility, the Editorial Note advised parents to "use caution when administering salicylates to treat children with viral illnesses, particularly chickenpox and influenza-like illnesses."

In October 1982, after CDC received a report of a fourth case-control study conducted in Michigan during the 1980–81 influenza season demonstrating a similar association, CDC convened a working group of expert consultants to review all four studies. The working group, which included pediatricians and epidemiologists as well as representatives of the Food and Drug Administration (FDA) and the American Academy of Pediatrics (AAP), reviewed the studies that had been completed and the

Reye Syndrome - Continued

many concerns expressed by those in the medical community, including consultants and representatives of the pharmaceutical industry. The working group supported CDC's original recommendation and stated that "until the nature of the association between salicylates and RS is clarified, the use of salicylates should be avoided, when possible, for children with varicella infections and during presumed influenza outbreaks" (3).

Soon after CDC made these recommendations, FDA conducted an independent audit and analysis of the data from the Ohio and Michigan studies. FDA then convened a scientific workshop to review the data, including analyses completed by FDA. Experts from the academic community, the pharmaceutical industry, and consumer organizations attended the meeting and had opportunities to present their independent analyses and concerns and to express their opinions regarding the studies. After an intensive review of all the concerns, the scientific working group concluded that the new analysis supported earlier evidence of the association between use of aspirin and increased risk for RS. As a result of this review process, in June 1982, the Surgeon General issued a recommendation advising "against the use of salicylate and salicylate-containing medications for children with influenza and chickenpox" (4).

Despite the numerous reviews by expert panels and intense scrutiny of the first four studies, many continued to express concerns about these studies, including industry representatives, the Office of Management and Budget (5), and the executive committee of the AAP, which issued a statement calling for further investigation. These concerns focused on the nature of the case-control studies and the many potential epidemiologic issues in such studies, including potential biases of selection and reporting as well as possible confounding (5). As a result of the concerns expressed by many groups, in December 1982, the Assistant Secretary of Health appointed a Public Health Service Task Force, comprised of representatives from CDC, FDA, and the National Institutes of Health, to assist in planning and conducting additional research on this issue. A decision about warning labels on packages of certain medications for children was deferred pending the results of this research (5).

The Public Health Service Task Force designed a new epidemiologic study to address the concerns that had been raised about the first four studies. A committee was convened by the Institute of Medicine to serve as an advisory board to review the protocol, monitor the study's progress, and review the analysis and results. Between February and May 1984, a pilot study, designed to test the methods for the main study of the relation between medication use and risk for RS, was undertaken. The pilot study, which involved 14 states and 33 pediatric tertiary-care centers, demonstrated a high odds ratio (16.1; lower 95% confidence limit=4.6) associated with the ingestion of aspirin during an antecedent respiratory or chickenpox illness and the development of Reye syndrome, consistent with the risks observed in previous studies. Evaluation of the epidemiologic issues raised concerning previous studies did not indicate that any of these issues could explain the observed association. Although in 1983 there had been no agreed-upon plans to publish the pilot study, the study was subsequently published in October 1985 (6) at the recommendation of the Institute of Medicine committee. In March 1986, FDA ruled that all over-the-counter aspirin and aspirincontaining products were required to be labeled with a warning about RS.*

^{*51} FR 8180-8182.

Reve Syndrome - Continued

Following completion of the pilot study, the main study of RS and medications was conducted during January 1985–May 1986. Although 70 pediatric tertiary-care centers throughout the United States participated in this study, including many that had previously reported the largest number of cases through CDC's national surveillance, only 33 cases of RS that met the study criteria were identified during the 17-month study period, which included two influenza seasons. However, the number of cases enrolled was fewer than had been expected based on prior experience and than had been specified by the original protocol (at least 100 cases), and the decision was made to discontinue the study because of the small number of cases identified, which reflected the declining incidence of RS that had been observed nationally during the preceding several years. In addition, in this study, as in the earlier studies, a high odds ratio was observed that could not be explained by any of the epidemiologic issues that the study had sought to address (7).

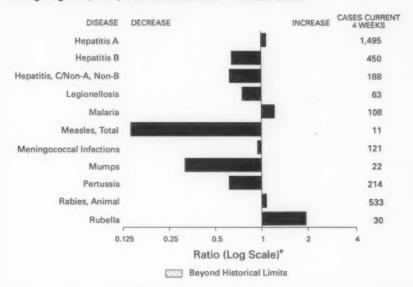
Although several years were required to address all the concerns about the initial studies, assessment of temporal trends in RS in the United States indicate that a dramatic decline in the incidence of this disease began to occur in the early 1980s soon after the initial and subsequent MMWR reports of these studies. This decline appeared to coincide with a decline in aspirin use among children that occurred as a result of the publicity surrounding these studies (8–10). The initial studies conducted during the early 1980s suggested that aspirin was administered to up to 70% of children with febrile respiratory illnesses. The national intervention involving the removal of a risk factor, aspirin use among children, was associated with a marked reduction in the incidence of this disease, providing the most convincing corroborating evidence for the association first reported in the case-control studies.

1997 Editorial Note by Eugene S Hurwitz, MD, Day Care Activities Coordinator, and Lawrence B Schonberger, MD, MPH, Assistant Director for Public Health, Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC.

References

- Reye RDK, Morgan G, Baral J. Encephalopathy and fatty degeneration of the viscera: a disease entity in childhood. Lancet 1963;2:749–52.
- Johnson GM, Scurletis TD, Carroll NB. A study of 16 fatal cases of encephalitis-like disease in North Carolina children. NC Med J 1963;24:464–73.
- CDC. National surveillance for Reye syndrome, 1981: update, Reye syndrome and salicylate usage. MMWR 1982;31:53–6,61.
- CDC. Surgeon General's advisory on the use of salicylates and Reye syndrome. MMWR 1982;31:289–90.
- Mortimer EA Jr. Reye's syndrome, salicylates, epidemiology, and public health policy [Editorial]. JAMA 1987;257:941.
- Hurwitz ES, Barrett MJ, Bregman D, et al. Public Health Service study of Reye's syndrome and medications: report of the pilot phase. N Engl J Med 1985;313:849–57.
- Hurwitz ES, Barrett MJ, Bregman D, et al. Public Health Service study of Reye's syndrome and medications: report of the main study. JAMA 1987;257:1905–1911.
- Arrowsmith JB, Kennedy DL, Kuritsky JN, Faich GA. National patterns of aspirin use and Reye syndrome reporting, United States, 1980 to 1985. Pediatrics 1987;79:858–63.
- Barrett MJ, Hurwitz ES, Schonberger LB, Rogers MF. Changing epidemiology of Reye syndrome in the United States. Pediatrics 1986;77:598–602.
- Remington PL, Rowley D, McGee H, Hall WN, Monto AS. Decreasing trends in Reye syndrome and aspirin use in Michigan, 1979 to 1984. Pediatrics 1986;77:93–8.

FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending August 9, 1997, with historical data - United States



^{*}Ratio of current 4-week total to mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

TABLE I. Summary — provisional cases of selected notifiable diseases, United States, cumulative, week ending August 9, 1997 (32nd Week)

	Cum. 1997		Cum. 1997
Anthrax Brucellosis Cholera Congenital rubella syndrome Cryptosporidiosis* Diphtheria Encephalitis: California* eastern equine* St. Louis* western equine* Hansen Disease Hantavirus pulmonary syndrome* Hemolytic uremic syndrome, post-diarrheal*	43 3 2 819 5 15 1 1 06 12 27	Plague Poliomyelitis, paralytic Paittacosis Rabies, human Rocky Mountain spotted fever (RMSF) Streptococal disease, invasive Group A Streptococal disease, invasive Group A Streptococal toxic-shock syndrome® Syphilis, congenital® Tetanus Toxic-shock syndrome Trichinosis Typhoid fever	26 2 208 988 23 190 26 74 6

no reported cases

^{*}Not notifiable in all states. *Not notifiable in all states. *Not notifiable in all states. *Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases (NCID). *Updated monthly to the Division of HIV/AIDS Prevention-Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP), last update July 29, 1997. *Updated from reports to the Division of STD Prevention, NCHSTP.

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending August 9, 1997, and August 10, 1996 (32nd Week)

						rrichia 157:H7			Hep	ntitia
		DS		mydia	NETSS	PHLIS ⁴	Gone	orrhea	C/N	
Reporting Area	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1997	Cum. 1997	Cum. 1996	Cum. 1987	Cum. 1996
UNITED STATES	34,732	40,090	256,848	260,894	1,184	672	161,292	191,236	1,915	2,171
NEW ENGLAND	1,478	1,713	10,579	10,388	106	46	3.587	3.893	43	61
Maine N.H.	36 19	29 50	590	547	8		36	29	43	61
Vt.	23	14	463 236	440 253	4 5	5	62	93	8	6
Mass.	533	870	4,377	4.022	65	40	32 1,352	1 215	2	16
R.I. Conn.	99	113	1,189	1,237	3	-	272	1,315	26 7	33
MID. ATLANTIC	768	637	3,724	3,889	21		1,833	2,105		-
Upstate N.Y.	11,041 1,754	11,284	35,732 N	39,879	57	19	21,212	25,243	219	179
N.Y. City	5,750	6,277	18,451	21,125	38	5	3,308	4,445	165	142
N.J.	2,211	2,219	5,508	7,512	11	8	8,224 4,074	9,552 4,988	-	3
Pa.	1,326	1,406	11,773	11,242	N	6	5,606	6,258	54	34
E.N. CENTRAL	2,441	3,208	35,704	52,640	228	123	22,646	34,851	325	324
Ind.	525 396	691 430	7,227	12,391	51	22	4,947	8,861	12	22
III.	899	1,396	9,500	5,810 15,144	40	10	3,632	3,754	10	7
Mich.	460	521	11,224	12,785	94	70	3,162 8,633	10,325 8,961	49	62
Wis.	161	170	5,260	6,510	N	21	2,272	2,950	254	233
W.N. CENTRAL	650	919	14,133	16,876	260	176	6,780	8,887	102	64
Minn. owa	128 75	169	U	3,128	132	119	U	1,381	3	1
Mo.	275	63 462	2,571 6,939	7,906	48	9	704	663	21	30
N. Dak.	9	11	473	559	29	36 6	4,573	5,203	65	15
S. Dak.	4	8	781	775	15	0	84	17 108	2	*
Nebr. Cans.	67 92	65 141	1,122	1,195	18		422	269	2	6
S. ATLANTIC			2,247	2,714	10	6	962	1,246	9	12
Del.	8,425 159	9,677	55,117 1,276	30,460	118	78	52,912	57,332	183	107
Vid.	1,075	1,145	4,256	1,148	11	3	720 7,926	875		*
D.C.	598	644	N	N	1	3	2,600	6,223 2,758	11	2
Va. W. Va.	719	646	7,023	6,535	N	18	4,795	5,800	18	8
N.C.	62 503	73 539	1,798	1,294	N	-	574	464	13	7
S.C.	484	498	7,461	Ü	37	22 5	11,062 6,651	11,263	38	30
Ga.	1,064	1,413	7,666	7,137	28		8,445	6,743 12,366	27 U	17
la.	3,761	4,530	14,295	14,346	33	27	10,139	10,840	76	43
S. CENTRAL	1,193	1,306	20,183	18,524	62	26	20,011	19,619	226	390
enn.	501	209 497	3,993 7,803	4,161	21		2,508	2,518	10	24
Ma.	285	364	4.888	8,001 5,133	31	26	6,557	6,928	156	296
Miss.	196	236	3,499	1,229	3		4,006	8,179 1,994	6 54	3 67
V.S. CENTRAL	3,615	3,953	34,760	33,665	38	5	21,774	23,186	272	-
Ark.	131	169	780	1,064	6	1	1,662	2,524	212	217
Okla.	622 188	927 166	5,671 4,542	4,264	4	3	5,301	4,568	131	129
ex.	2,674	2,691	23,767	4,692 23,645	26	1	2,868 11,943	2,906	6	1
MOUNTAIN	1,022	1,189	14,175	15,419	136	70		13,188	135	83
Mont.	26	22	644	768	14	78	4,414	4,841	249	379
daho Vyo.	34	25	890	944	15	8	68	17 67	14 34	11
colo.	13 250	333	345 1.896	389	7		35	22	104	120
I. Mex.	104	111	2,081	1,290	54	39	1,249	1,090	26	35
Vriz.	255	342	5,864	6,803	N	21	706 1,750	511 2,343	33	47
itah iev.	82	114	954	919	33	*	140	183	23	44 17
ACIFIC	258	239	1,501	1,813	8	6	439	608	12	17
Vash.	4,867 421	6,840	36,465	41,043	179	118	7,956	13,384	296	450
reg.	188	445 311	5,534 2,971	6,092	45 50	22	1,165	1,319	19	36
alif.	4,187	5,946	26,147	33,191	76	58 31	461 5,819	11 504	2	*
Jaska	36	16	864	692	8	1	235	11,504 266	177	285
lawaii	35	122	949	1,068	N	6	276	295	98	127
iuam R.	1 100	4 227	31	248	N		3	42		6
LL.	1,199	1,337 16	N	U	28	U	392	389	74	113
lmer. Samoa	*	10	14	N	N	U				
.N.M.I.										

N: Not notifiable U: Unavailable : no reported cases C.N.M.L.: Commonwealth of Northern Mariana Islands

**Updated monthly to the Division of HIV/AIDS Prevention—Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention,

Islanda Electronic Telecommunications System for Surveillance.

Public Health Laboratory Information System.

TABLE II. (Cont'd.) Provisional cases of selected notifiable diseases, United States, weeks ending August 9, 1997, and August 10, 1996 (32nd Week)

		ellosis	Ly: Dise		Mai	laría		hilis Secondary)	Tuber	culosis	Rabies
Reporting Area	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1957	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997
UNITED STATES	508	515	3,462	6,938	924	868	4 : 28	7,401	10,341	11,372	4.549
NEW ENGLAND	38	27	769	1,936	41	33	96	108	264	253	
Maine	2	1	8	14	1	6	-	100	11	16	130
N.H. Vt.	4 7	1	9	28	1	1	-	1	10	8	25
Mass.	9	14	129	10 81	2	2		-	4	1	90
R.I.	5	7	195	202	18 5	11	45 2	50	151	112	142
Conn.	11	N	422	1,601	14	8	49	56	19	24 92	15
MID. ATLANTIC	90	114	2,042	4.144	231	267	229				280
Jpstate N.Y.	24	36	637	1,994	42	53	21	316 49	1,915	2,053	931
N.Y. City N.J.	12	8	27	216	122	151	53	95	986	1,083	693
Pa.	51	61	670 708	902	49	47	88	108	394	442	102
E.N. CENTRAL	156			1,032	18	16	67	64	294	285	136
Ohio	78	170 54	49 32	280	82 12	109	404	1,143	1,017	1,216	96
nd.	27	36	15	14	8	9	119	436 146	180	174	65
Hf.	5	23	2	8	29	56	39	314	90 517	111 665	8
Mich. Wis.	39	30		6	25	22	93	122	157	204	13
		27	U	239	8	13	63	125	73	62	2
W.N. CENTRAL Minn.	44	25	48	93	31	24	93	227	327	302	296
owa	12	3	32 5	18 13	10	7	U	26	86	70	29
Mo.	11	5	7	34	10	2 8	61	15 162	38	43	107
N. Dak.	2	*	*	-	2	1	01	102	132	126	15
S. Dak. Nebr.	2	2	1	*		*			7	14	40
Cans.	12	9 2	2	2	1	2	5	8	14	13	1
S. ATLANTIC				26	2	4	21	16	42	33	60
Del.	72 6	71	349 27	322 122	196	144	2,001	2,363	1,973	2,071	1,863
Md.	17	13	245	113	57	3	16 524	23 412	11	28	43
D.C.	3	6	7	1	10	7	77	90	192 59	181	339
N. Va.	14	13	24	24	43	24	152	279	194	178	374
V.C.	N 9	N 6	21	9		3	3	2	37	40	58
S.C.	3	4	21	43	10	15	453	643	251	297	556
Ga.		3	1	1	21	16	237 342	254 421	199 370	208 387	103
Fla.	20	17	20	6	43	28	197	239	660	670	186
E.S. CENTRAL	32	29	45	49	20	22	1,098	1,612	760	867	199
(y. Tenn.	22	14	6	15	4	6	91	83	112	148	21
Ala.	2	3	24	16 5	6 7	9	485	530	245	299	123
Aiss.	4	10	11	13	3	3	277 245	355 644	251	269	55
V.S. CENTRAL	13	16	48	70	10	22	-		152	151	
Ark.	*	1	11	19	2	22	683 68	1,163 165	1,433	1,378	223
.a.	2	1	2	1	8	2	230	334	136	118	27
Okla. Tex.	3	10	9	5		*	73	123	109	110	70
MOUNTAIN			26	45	-	20	312	541	1,064	1,141	124
Aont.	32	31	12	4	50	35	96	94	299	385	92
daho	2		2		2	5	*	-	7	14	26
Vyo.	1	3	2	3	2	3	-	2 2	8 2	6	20
Colo.	8	7	4		24	16	7	24	57	51	20
I. Mex. kriz.	2	12	1	*	7	1	8	4	16	56	9
Itah	6	2	1	1	7	4	70	49	150	150	34
lev.	4	5	2		5	2	7	11	13 46	34	2
ACIFIC	31	32	100	40	263	212				70	3
Vash.	6	3	5	5	13	12	128	375 7	2,353	2,847	168
reg.	2.	-	11	-	15		5		103	162	7
alif. Jaska	24	27	84	34	230	191	114	366	1,900	2,523	142
awaii	i	1		1	3 2	3	1	-	52	50	19
uam		1			2	6	1	2	108	112	*
R.					4	i	154	3	5	55	-
LL,					-	1	154	147	129	105	41
mer. Samoa	*	*	*			*					
N.M.I.		*					9	1	2		

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending August 9, 1997, and August 10, 1996 (32nd Week)

		uenzae,	H	lepatitis (\	/iral), by ty	pe			Measi	es (Ruber	ola)	
	Cum.	sive		A	_	В	India	enous	Imp	orted [†]	To	otal
Reporting Area	1997°	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	1997	Cum. 1997	1997	Cum. 1997	Cum. 1997	Cum. 1996
UNITED STATES	687	692	16,573	16,101	5,254	5,785	1	57	1	40	97	
NEW ENGLAND	37	24	398	202	94	135		10		6		393
Maine N.H.	3 5	10	45	13	6	2				1	16	13
Vt.	3	10	21	9	7 5	8		1	-	-	1	
Mass.	22	12	151	103	37	10 45	-	9	-	-	-	2
R.I. Conn.	2	1	95	9	11	7			-	4	13	10
MID. ATLANTIC	2		78	64	28	63	*		*	1	1	1
Upstate N.Y.	78 14	149 37	1,216	1,132	782	918	1	14	1	7	21	33
N.Y. City	22	40	451	260 352	168 267	219 333	-	2	*	3	5	7
N.J. Pa.	32	38	193	230	155	180		5	-	2	7	11
	10	34	393	290	192	186	1	6	1	2	8	12
E.N. CENTRAL Ohio	112	122	1,569	1,528	533	679		5		3	8	16
Ind.	66 11	70	218 190	536	55	85	*		+	-		2
III.	24	32	338	191 395	64 124	92 203	-		+	:	-	
Mich. Wis.	10	8	730	271	266	240	-	5	-	1 2	6 2	3
	1	5	93	135	24	59	-	-		-	2	9
W.N. CENTRAL	37	30	1,271	1,333	310	296		9	-	3	12	18
lowa	25 5	18	111	76	23	35				3	3	16
Mo.	3	6	235 651	227 682	33 218	40 176	*		*	*	-	
N. Dak.			10	28	3	1/0	-	1			1	1
S. Dak, Nebr,	2	1	17	39	1	2		8	-		8	
Kans.	1	1	61 186	97 184	10	21	*	*	-	-		
S. ATLANTIC	122	129	1,068	683	-	22	*	*		*	-	1
Del.	*	2	22	9	777	789 6		3	7	8	11	8
Md. D.C.	46	44	161	122	110	106	-			2	2	1
Va.	2 9	5	16	20	25	26				1	1	1
N. Va.	3	6	137	98 12	79	90	*		-	1	1	2
N.C.	17	20	121	87	161	227		1	*	1		
S.C. 3a.	23	4	71	37	62	49	-			1	2	2
la.	19	30 12	230 304	60 238	83 244	8	*	*	*	1	1	1
E.S. CENTRAL	37	20	403			263		2	*	1	3	1
(y.	5	5	51	911 26	419 25	508		*	*	*	-	
enn.	24	8	253	608	280	286				*		*
Ala. Miss.	8	6	59	124	41	40		*				-
W.S. CENTRAL		1	40	153	73	135	-	*		*		
Ark.	33	30	3,530 160	3,236	701	689	-	3	-	4	7	22
.8.	7	3	139	294 106	41 94	52 75	-	*	*	-		-
Okla. Tex.	22	23	993	1,385	24	24		*				*
	3	4	2,238	1,451	542	538		3		4	7	22
AOUNTAIN Aont.	73	38	2,729	2,722	579	711		7		1	8	149
daho	1	1	57 85	80 148	6	7	*		-		-	143
Vyo.	2	-	21	25	17	67 30	-	*	*		*	1
iolo. I. Mex.	10	11	277	275	111	80			*	*	*	
Ariz.	8 28	9	1,398	273	187	245	*					10
Ptah	3	5	404	1,061	130 65	166	*	5	*	*	5	8
lev.	21		273	252	39	52		1		1	1	118
ACIFIC	158	150	4,389	4,354	1,059	1,060		6	-		2	5
Vash. Freg.	3	2	328	320	48	59		1		8	14	134
rreg. alif.	26 119	142	240	2040	64	-			*			38
Jaska	3	4	3,717	3,948	925	987	*	3		7	10	31
lawaii	7	2	80	54	8	6		2	-	1	-	63
uam	*			6	1		**	2		1	3	2
R.	*	1	201	132	914	636	U		U	*	*	-
II. mer. Samoa	*	*	*	26		25	U		U	-	~	2
N.M.L	6	10	1	i	34	5	U		U	*		
l: Not notifiable		ailable		,	34	D	U	1	U		1	~

N: Not notifiable U: Unavailable -: no reported cases

^{*}Of 149 cases among children aged <5 years, serotype was reported for 79 and of those, 31 were type b. [†]For imported messles, cases include only those resulting from importation from other countries.

TABLE III. (Cont'd.) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending August 9, 1997, and August 10, 1996 (32nd Week)

		ococcal ease		Mumps			Portussis			Rubella	
Reporting Area	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum 1996
UNITED STATES	2,198	2,078	6	365	441	54	2,960	2,598		121	202
NEW ENGLAND	139	94		8	1	2	572	589		121	24
Maine N.H.	15	10	*			-	6	21			24
N.H. Vt.	13	3			-	*	66	40	*		
Mass.	70	36		2	1	2	181 295	16 507			2
R.I.	12	10	*	5		-	12	307			20
Conn.	26	32	*	1		*	12	5			2
MID. ATLANTIC Upstate N.Y.	203 50	235 60	-	35	56	4	201	172		24	10
N.Y. City	37	35		7 3	17 13	1	59 52	85 22	*	1	4
N.J.	44	52			2		5	11		23	4 2
Pa.	72	88	*	25	24	3	85	54	-		-
E.N. CENTRAL Ohio	310	314		40	93	3	233	325		4	3
ind.	121 34	116 45	*	18	32	3	95	110	*		
111.	93	87		7	5 18	-	35 37	19		i	i
Mich.	37	31		9	37		31	27	-		2
Wis.	25	35	*	*	1		35	100		3	-
W.N. CENTRAL Minn,	162	173		13	11	10	190	125			-
lowa	24 38	23 37		5	3	10	130	89	*		-
Mo.	74	64		6	1 4		19 27	3 18	*		-
N. Dak.	1	3		-	2		2	1			
S. Dak. Nebr.	6	9	*		*	*	3	3			-
Kans.	15	21		2	1	-	5	5	*		*
S. ATLANTIC	393	337	1	51	69						
Del.	5	2		21	03	8	299	267 17		62	89
Md. D.C.	36	39		4	24	5	92	103			
Va.	37	5 35	î	9	10	*	3				1
W. Va.	14	13		9	10	2	34	27		1	2
N.C.	74	58		7	14	*	80	47		50	75
S.C. Ga.	44 75	100		10	5 2	3	14	18		9	1
Fla.	107	44		16	14	-	9 62	13	*	2	10
E.S. CENTRAL	172	154		18	18	1	67	164			
Ky.	37	20	*	3			15	131		-	2
Tenn. Ala.	67 52	46 50	*	3	1	1	27	15		-	-
Miss.	16	38		6	3 14		16	11 7	*		2
W.S. CENTRAL	218	234		33	30				•		N
Ark.	25	27		33	1	-	73 13	77		3	7
La. Okla.	45	45	-	11	11		13	6			1
Tex.	24 124	139	*	22	18	*	14	8	*		
MOUNTAIN	130	127		-			33	60	*	3	6
Wont.	8	6	1	49	18	18	835 16	261 13		5	6
daho	8	20		2	-	6	537	76		1	2
Myo. Colo.	1 36	3		1	-		6	2			
V. Mex.	21	22	N	3 N	3 N	3	178	81	*		2
Ariz.	35	30		31	1	6	53 23	36 15	-	4	1
Itah	11	12	-	6	3	-	10	10	-		
Nev.	10	13	1	6	11	2	12	28			1
ACIFIC Wash.	471	410	4	118	146	8	490	618	*	23	61
Oreg.	59 94	65	1 N	14 N	18 N	8	224	227	*	5	12
Calif.	313	337		86	104		17 236	371	-	10	46
Maska	1	5	-	2	2		2	1			40
favaii	4	3	3	16	21	*	11	19	-	8	3
Buam R.		4	U	1	4	U		-	U		
/.l.	9	10	ú	5	1		-	2	*	*	*
Amer. Samoa	4		Ü		1	U			Ü	*	-
I.N.M.L			U	4	-	Ŭ			ŭ		

N: Not notifiable

U: Unavailable

-: no reported cases

TABLE IV. Deaths in 122 U.S. cities,* week ending August 9, 1997 (32nd Week)

	A	II Cau	ses, By	Age (Y	ears)		P&d*		A	III Cau	ses, By	Age (Y	ears)		P&
Reporting Area	All Ages	>65	45-64	25-44	1-24	<1	Total	Reporting Area	All Ages	>65	45-64	25-44	1-24	<1	Tot
NEW ENGLAND Boston, Mass.	476 162	315 96	95 32	41 19	15 11	10	25 9	S. ATLANTIC Atlanta, Ga.	1,200 143	713 88	280 35	131 10	44 8	27 2	41
Bridgeport, Conn. Cambridge, Mass.	14	13		1	*		1	Baltimore, Md. Charlotte, N.C.	197	107 55	51	28	6	4 2	1:
all River, Mass.	30	24	4	1		1	-	Jacksonville, Fla.	126	84	21	13	6	2	
lartford, Conn.	18	14	3		1	-	-	Miami, Fla.	109	56	25	19	5	2	
owell, Mass. ynn, Mass.	25 15	20	4	1 4			2 2	Norfolk, Va. Richmond, Va.	46 86	28 39	9	10	2	5	
lew Bedford, Mass	. 20	16	4		*			Savannah, Ga.	48	32	10	2	3	1	
lew Haven, Conn.	30 47	19 26		3 5	2	3	1	St. Petersburg, Fla.	164	104	12 37	18	1 2	2	
rovidence, R.I. iomerville, Mass.	5	3		5	- 2	1	1	Tampa, Fla. Washington, D.C.	106	61	27	6	8	4	1
pringfield, Mass.	35	26	8	1		*	3	Wilmington, Del.	24	18		6		*	
Vaterbury, Conn. Vorcester, Mass.	23 50	14 35		2 4	1	1	6	E.S. CENTRAL	798	546	140	67	26	17	5
				-				Birmingham, Ala.	167	126	21	11	6	1	2
Albany, N.Y.	2,082	1,387	406	208	46	32	87	Chattanooga, Tenn. Knoxville, Tenn.	82 121	54 90	14 18	9	4	3	
Allentown, Pa.	19	15	3	1				Lexington, Ky.	46	31	8	4	-	3	
Buffalo, N.Y.	58	45		1	-	2	1	Memphis, Tenn.	146	90	34	16	4	2	
Camden, N.J. Elizabeth, N.J.	32 27	18 15		5 2	1	2	*	Mobile, Ala. Montgomery, Ala.	97 21	69 14	15	6	4	3	
Frie, Pa.	40	34		2		-	1	Nashville, Tenn.	118	72	25	14	3	4	
Jersey City, N.J.	33	24		3	1		.1	W.S. CENTRAL	1,451	895	298	140	62	56	7
New York City, N.Y. Newark, N.J.	1,101	740		109	23	14	44 2	Austin, Tex.	73	48	15	5	1	4	,
aterson, N.J.	18	9		2	3		-	Baton Rouge, La.	36	25	6	4	-	1	
hiladelphia, Pa.	300	176		42	5	6	17	Corpus Christi, Tex. Dallas, Tex.	60 214	39 130	14	19	11	16	
ittsburgh, Pa.§ Reading, Pa.	42	30		3	*	3	3	El Paso, Tex.	91	59	20	8	1		
Rochester, N.Y.	137	95		10	4	2	6	Ft. Worth, Tex.	83	51	17	8	5	2	1
Schenectady, N.Y.	24	21	3		-	-	1	Houston, Tex. Little Rock, Ark.	343	185 54	89 17	42	18	9	2
Scranton, Pa. Syracuse, N.Y.	25 70	20 55		3	2	2	6	New Orleans, La.	89	43	11	15	15	3 2 9 2 5 7	
Trenton, N.J.	29	25		3	-	-	5	San Antonio, Tex.	195	135	34	17	2	7	1
Utica, N.Y.	14	10	2	2				Shreveport, La. Tulsa, Okla.	65 118	53 73	7	5	5	2	
Yonkers, N.Y.	U	U		U	U	U		THE PARTY OF THE P			146				
E.N. CENTRAL	2,088	1,415		160	49	32		MOUNTAIN Albuquerque, N.M.	837 65	555 43	140	72 5	42	21	4
Akron, Ohio Canton, Ohio	39	31		1		1		Boise, Idaho	31	22	7	1	1		
Chicago, III.	431	253	117	41	15	4	30	Colo. Springs, Colo		31	8	2	4	1	
Cincinnati, Ohio Cleveland, Ohio	129	80		14	7 2	3	14	Denver, Colo. Las Vegas, Nev.	152 138	98	28	12	10	4 2	1
Columbus, Ohio	154 227	100	44	14	4	1		Ogden, Utah	30	23	3	4	*		
Dayton, Ohio	120	88	25	7	*		11	Phoenix, Ariz.	154	90	26	23	10	4	
Detroit, Mich. Evansville, Ind.	181	110	40	22	4	5	5	Pueblo, Colo. Salt Lake City, Utah	32	19 63	6	6	3	2	
Fort Wayne, Ind.	55	36		4	3			Tucson, Ariz.	101	73	16	6	3	5	
Gary, Ind.	12	3	2	3	4			PACIFIC	2,075	1,440	378	143	70	44	13
Grand Rapids, Mich	1. 57 185	128		13	1 4	1 2		Berkeley, Calif.	14	11	2 7	1		-	
Indianapolis, Ind. Lansing, Mich.	38	28		2	**	-		Fresno, Calif.	50	30		5	6	2	
Milwaukee, Wis.	101	70	22	5	*	4	8	Glendale, Calif. Honolulu, Hawaii	26 75	21 53	12	7	-	3	
Peoria, III. Rockford, III.	31 55	23		3	2	1		Long Beach, Calif.	70	41	17	8	4		
South Bend, Ind.	34	29		3	-			Los Angeles, Calif.	710	484	135	56	19	16	
Toledo, Ohio	90	63	14	7	2	4		Pasadena, Calif. Portland, Oreg.	200	129	45	14	12	*	
Youngstown, Ohio	55	50) 4	1				Sacramento, Calif.	203	141	40	11	7	4	
W.N. CENTRAL	624	443		35	11	16		San Diego, Calif.	127 f. 90	88		8	7	5	
Des Moines, Iowa	U 25	27	3	U	U	U	U	San Francisco, Cali San Jose, Calif.	172	58 126		6	4	5	
Duluth, Minn. Kansas City, Kans.	27	15				1		Santa Cruz, Calif.	29	24	5			*	
Kansas City, Mo.	78	5	1 10		4			Seattle, Wash.	141	102	19	11	7	2	
Lincoln, Nebr.	36	20			1	2 2 2	1	Spokane, Wash. Tacoma, Wash.	46 88	36 68		5	1	2	
Minneapolis, Minn. Omaha, Nebr.	166				2	4	7 3								-
St. Louis, Mo.	101	71	3 16	5	1	1		TOTAL	11,631	7,709	2,287	997	365	255	6
St. Paul, Minn. Wichita, Kans.	54 58			1 4	1	4									

U: Unavailable : no reported cases

*Mortality data in this table are voluntarily reported from 122 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

included.

Presumonia and influenza.

Because of changes in reporting methods in this Pennsylvania city, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

Total includes unknown ages.

Contributors to the Production of the MMWR (Weekly)

Weekly Notifiable Disease Morbidity Data and 122 Cities Mortality Data

Denise Koo, M.D., M.P.H.

State Support Team

Robert Fagan

Karl A. Brendel

Siobhan Gilchrist, M.P.H.

Harry Holden

Gerald Jones

Felicia Perry

Carol A. Worsham

CDC Operations Team

Carol M. Knowles

Deborah A. Adams

Willie J. Anderson

Christine R. Burgess

Patsy A. Hall

Myra A. Montalbano

Angela Trosclair, M.S.

Desktop Publishing and Graphics Support

Morie M. Higgins

Peter M. Jenkins

The Morbidity and Mortality Weekly Report (MMWR) Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to listserv@listserv.cdc.gov. The body content should read SUBscribe mmwr-toc. Electronic copy also is available from CDC's World-Wide Web server at http://www.cdc.gov/ or from CDC's file transfer protocol server at ftp.cdc.gov. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

Data in the weekly MMWR are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the MMWR Series, including material to be considered for publication, to: Editor, MMWR Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

All material in the MMWR Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

- Director, Centers for Disease Control and Prevention David Satcher, M.D., Ph.D.
- Deputy Director, Centers for Disease Control and Prevention Claire V. Broome, M.D.
- Director, Epidemiology Program Office Stephen B. Thacker, M.D., M.Sc. Editor, MMWR Series Richard A. Goodman, M.D., M.P.H.
- Acting Editor, MMWR (weekly) John W. Ward, M.D. Managing Editor, MMWR (weekly) Karen L. Foster, M.A. Writers-Editors, MMWR (weekly) David C. Johnson Darlene D. Rumph Person Teresa F. Rutledge Caran R. Wilbanks

PUNCA ZOMZO COMMO HEN DZDM NOLD DODGO OH HO DIDAM 040

SNO

1 0 DEP DEP

Un U

Laj HIMERO minim FONDE 一次多 PRIOR WHZHO r~

-

☆U.S. Government Printing Office: 1997-532-228/67021 Region IV

Address Correction Requested Official Business

Atlanta, Georgia 30333 and Prevention (CDC)

HEALTH AND HUMAN SERVICES DEPARTMENT OF Centers for Disease Control Public Health Service

Permit No. G-284 PHS/CDC POSTAGE & FEES PAID

FIRST-CLASS MAIL

Redistribution using permit imprint is illegal

